

The Effects of Sleep Deprivation on Dissociation and Profiles of Mood, and Its Association with Biochemical Changes

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ABSTRACT

Introduction: Sleep deprivation is a method, which has been used in order to comprehend the functions of sleep both in healthy individuals and for the patients of depression with in treatment, for a long time. The objective of our present study is to examine the relation between hormonal values, which are known for being related to the effects of these said changes determined in the mood, dissociation and thought suppression in healthy individuals after one night of sleep deprivation implementation.

Methods: One night sleep deprivation was performed on a total of thirty-two healthy volunteers (16 males and 16 females) who were included in the study. Blood samples were taken from the individuals before and after sleep deprivation implementation in order to determine cortisol, dehydroepiandrosterone-sulfate (DHEA-S) and Thyroid Functions' Levels tests. In order to evaluate the effects of the sleep deprivation on moods, "White Bear Suppression Inventory (WBSI)" has been conducted, with an aim of evaluating thought suppression, "Profile of Mood States (POMS)", "Dissociative Experiences Scale (DES)" with a purpose of realizing any dissociation tendency.

Results: On the individuals who have been implemented for sleep deprivation, a decrease on depression and vigor-activity sub-scales values was

detected, and an increase was determined on fatigue sub-scales values of "POMS". While the values of DES were found to have been statistically increased after sleep deprivation, also a significant decrease was determined on WBSI values. Even if there hasn't been any significant statistical change determined on cortisol levels after sleep deprivation, yet there had been some significant changes detected on Thyroid Stimulating Hormone (TSH), fT3, fT4, and DHEA-S levels. Decrease in "POMS" depression sub-scale values and increase on fatigue sub-scale values were determined on the individuals whose sT4 levels were found to be increased significantly in statistic manner after the sleep deprivation.

Conclusion: According to the results of our study, sleep deprivation for one night was determined to cause decrease on depressive mood, increase on dissociative symptoms and to lower the tendency of suppressing the unwanted thoughts, consciously. The fact of being obtained lower depression values, on the individuals with the increased DHEA-S levels after the sleep deprivation meets with the information claiming that the high DHEA-S levels may be deemed as protectors against the negative effects of the stress.

Keywords: Sleep deprivation, dissociation, mood, biochemical changes

INTRODUCTION

Sleep is a vital process controlled at and affecting every level of a biological structure. Healthy maintenance of thermoregulation, endocrine system, metabolic functioning, and homeostatic system, including immune system, can be impaired because of sleep disorders (1). Sleep deprivation is applied for many years both in healthy individuals to understand functions of sleep and in depressive disorders for treatment purposes (2). In the meta-analysis of Wu and Bunney, it is reported that 50%–60% of the depressive patients experienced a rapid but temporary mood improvement following sleep deprivation (3). Similarly, sleep deprivation in healthy individuals also resulted in a significant improvement in terms of depressive complaints, depending on individual and psychobiological differences in sleep-wake rhythms (4).

Discussions on the assertion that dissociative experiences are caused by disorders in sleep-weak rhythm are going on to explain the relation between sleep and dissociation. The relation between nightmare disorder, which is a parasomnia and dissociative experiences, and the role of childhood traumas in this relation has been demonstrated before (5). Although it has been demonstrated through subjective assessments that individuals having more sleep disorders go through dissociative experiences such as depersonalization, derealization, and absorption, the neurobiological evidence of changes in dissociative symptoms following sleep deprivation are still not sufficient (6).

Studies researching the effects of sleep loss on cognitive performance show that sleeplessness impairs performance skills, decreases attention and wakefulness, and has a negative effect on learning ability (7,8). Impaired instant recall function, which is included in the category of short-term memory, is a typical finding of sleep deprivation studies (8). It is projected that it takes place because of ignoring for-



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mer elements when new elements are presented. In addition, reasoning and concentration abilities also decrease because of mood changes (7,9).

The hypothalamic-pituitary-adrenal axis (HPA) acts as a regulator of a behavioral and autonomic response to stress. Recent studies show that sleep deprivation acts as a stressor and leads to a moderate activation in the HPA axis and an increase in plasma glucocorticoid levels (10). Dehydroepiandrosterone-sulfate (DHEA-S), which is another hormone regulated by circadian rhythm, has significant anti-glucocorticoid effects through the inhibition of enzyme activities stimulated by glucocorticoids, and it is asserted that it can prevent destructive effects of hypercortisolemia in case of acute stress (11). Dehydroepiandrosterone (DHEA) and DHEA-S have neuroprotective effects; they increase neuronal plasticity and excitability. There is an interrelation between sleep, energy metabolism and thyroid functions. Thyroid stimulating hormone (TSH) release has a significant pulsatile profile. Sleep causes a fracture in the HPA axis, and sleep deprivation leads to a powerful increase in TSH and related thyroid hormones, with an increase in thyroid releasing hormones (TRH) (12). On the other hand, the interrelation between sleep deprivation, mood states and hormonal changes are not sufficiently clear. In our modern world today, healthy individuals have a limited sleep cycle, which differs from their natural biological rhythms, because of social activities. Besides, the shift system also causes sleep deprivation and it is described as chronic sleep deprivation (13).

This study is intended to discuss the relation between changes in mood states, dissociation and thinking processes that may appear in healthy individuals following one night sleeplessness and hormonal changes that have a circadian rhythm and are strongly.

METHODS

Participants

Thirty-two voluntary healthy university students consisting of 16 males and 16 females have been included in this study. The inclusion criteria for the study have been defined as follows: age >18 years, having no physical or psychiatric disorder, receiving no psychiatric diagnosis of Axis I or Axis II before the study period, using no drugs, alcohol, or substances that are known to affect sleep within the last 15 days and during the study period, and not working in a shift system.

Blood samples have been taken from the individuals meeting the study participation criteria for cortisol, DHEA-S, and thyroid function tests at 08:00 in the morning before and after sleep deprivation. The Dissociative Experiences Scale (DES), the Profile of Mood States (POMS), and the White Bear Suppression Inventory (WBSI), all of which are a self-report measure, have been applied.

Measures

Dissociative experiences scale (DES): It is a self-report measure consisting of 28 questions used to screen dissociative experiences and measure their severity. Each item is scored between 0 and 100. The average score is obtained by dividing the sum of these scores by 28. Individuals whose overall score is equal to or above 30 are likely to have a dissociative disorder. The validity and reliability studies have been conducted by Yargıç et al. (14).

White bear suppression inventory (WBSI): It is a self-report measure developed by Wegner and Zanakos and intended to evaluate the tendency to consciously suppress unwanted thoughts. It is also used

(OCD), and psychiatric disorders such as depression. It totally consists of 15 items. Each item is scored between 1 and 5 depending on the answer. The total score of answers to all the items gives the total measure score. The total score can vary from 15 to 75. High scores indicate that there is a stronger tendency or potential to consciously suppress unwanted thoughts. The validity and reliability studies in Turkey have been conducted by Ağargün et al. (15).

Profile of mood states (POMS): It has been developed to quickly and reliably define and evaluate situational and short-term changes in mood states (16). It is used to evaluate the effects of psychotherapies and medications, measure the efficiency of sleep deprivation and other similar applications, and survey mood state changes in experimental and clinical studies. As the result of random ordering of the questions forming the measure, certain questions are categorized under 6 individual mood states. These are as follows: "tension-anxiety," "depression-dejection," "anger-hostility," "vigor-activity," "fatigue-inertia," and "confusion-bewilderment." The total POMS score is obtained by deducting the subscale score of "vigor-activity" from the sum of the other five subscale scores. If these five subscale and total scale scores are high, it indicates a higher disorder in mood states. The validity and reliability studies for the scale have been published by Selvi et al. (17) in Turkey.

Procedure

Psychiatric disorder in the individuals whose approvals have been taken and who have met the study participation criteria have been ruled out through the Structured Clinical Interview for DSM Disorders (SCID) evaluation. It was planned to put 32 individuals consisting of 16 males and 16 females through sleep deprivation. The individuals have been accepted to a clinic in 3-people groups each week for the application of total sleep deprivation. Individuals put through total sleep deprivation have been deprived of sleep from 22:00 at night to 07:00 in the morning and were continuously observed by the doctor in charge and the allied health personnel to prevent them from sleeping.

Before the sleep deprivation application, blood samples have been taken from all the subjects at 08:00 in the morning for cortisol, DHEA-S, and Thyroid Function Tests (TFT); POMS, DES, and WBSI scales have been applied. Blood sampling at 08:00 in the morning and scale applications have been repeated following sleep deprivation. Blood samples have been centrifuged at 5000 rpm for 10 min in the biochemistry laboratory to separate serum. Immunoassay kits compatible with fT_3 , fT_4 , and TSH Architect I 2000 and Immulite 2000 for cortisol and DHEA-S and proper immunoassay kits have been used.

Statistical Analysis

Because all the individuals have fully met the study participation criteria, all of them have been put through statistical evaluation. Age, sex, marital status, all POMS values and sum of POMS subscale scores (six subscales), total POMS value, DES, WBSI, and cortisol, DHEA-S, fT_3 , fT_4 , and TSH values before and after sleep deprivation have been entered into the statistical package in SPSS 16.0. Pearson correlation coefficient has been used to analyze the correlation of POMS, DES, WBSI, and cortisol, DHEA-S, fT_3 , fT_4 , and TSH values before and after sleep deprivation. T-test has been applied to dependent groups for group comparison purposes. $p < 0.05$ has been considered statistically significant.

RESULTS

The average age of the individuals was 24 years for males and 27 years for females. When POMS subscales were evaluated before and after sleep deprivation, it has been observed that depression scores show a significant

decrease after sleep deprivation, whereas fatigue scores increase significantly and vigor-activity scores decrease significantly ($p < 0.05$). It has been determined that changes in tension-anxiety, anger-hostility, and confusion subscales and overall POMS scores after sleep deprivation are not statistically significant. A significant decrease has been observed in DES and WBSI scores after sleep deprivation ($p < 0.05$) (Table 1).

The evaluation of biochemical parameters following sleep deprivation showed a significant increase in TSH, T_4 , T_3 , and DHEA-S levels following sleep deprivation ($p < 0.05$). No statistically significant relation has been found between POMS subscales of "tension-anxiety," "anger-hostility," and "confusion-bewilderment," overall POMS scores, cortisol levels, and DHEA-S/cortisol rates before and after sleep deprivation (Table 1).

Correlations of POMS subscales, overall POMS scores, and DES and WBSI scales have been evaluated with biochemical parameters. TSH indicated a positive correlation with "tension-anxiety," "anger-hostility," "fatigue-inertia," and "confusion-bewilderment" subscales and overall POMS score and a negative correlation with "depression-dejection" and "vigor-activity" scores; however, these results were not statistically significant.

Free T_4 (T_4) indicated a positive correlation with "anger-hostility," "confusion-bewilderment," "fatigue-inertia," and "vigor-activity" subscales and DES scores and a negative correlation with "depression-dejection," "tension-anxiety," and WBSI. Of these correlations, the correlations with "depression-dejection" and "fatigue-inertia" subscales were statistically significant.

Free T_3 (T_3) indicated a positive correlation with "depression-dejection," "tension-anxiety," "anger-hostility," "fatigue-inertia," and "confusion-bewilderment" subscales and overall POMS score and a negative correlation with "vigor-activity," DES, and WBSI, although it was not statistically significant (Table 2).

Cortisol indicated a positive correlation with the "confusion-bewilderment" subscale only. This correlation was not statistically significant (Table 2).

DHEA-S had a positive correlation with "confusion-bewilderment," "anger-hostility," "fatigue-inertia," DES, and WBSI and a negative correlation with "depression-dejection," "tension-anxiety," and "vigor-activity" subscale scores and overall POMS score. Of these, the correlations with "depression-dejection," "fatigue-inertia," and DES were significantly meaningful ($p < 0.001$) (Table 2).

In addition, correlations of changes in POMS subscales following sleep deprivation have been identified. "Tension-anxiety," "depression-dejection," and "anger-hostility" subscales showed a positive correlation, whereas the "vigor-activity" subscale showed a negative correlation. The "Depression-dejection" subscale had a positive correlation with "tension-anxiety," "anger-hostility," and "fatigue-inertia" subscales and a negative correlation with "vigor-activity." The "Anger-hostility" subscale showed a positive correlation with "tension-anxiety" and "depression-dejection" subscales in addition to the "fatigue-inertia" subscale and a negative correlation with the "vigor-activity" subscale. A negative correlation has been found between "fatigue-inertia" and "vigor-activity" subscales. Evaluation of the correlation of biochemical parameters with each other following sleep deprivation indicated a positive correlation between changes in TSH levels that appeared following sleep deprivation and changes in T_3 levels and these discrepancies were statistically significant ($p < 0.001$). The correlation between T_4 values and DHEA-S values was positive and sta-

Table 1. Changes in mood, dissociation, thought suppression and biochemical parameters of healthy individuals before and after total sleep deprivation according to the POMS, DES and WBSI evaluations

	Before sleep deprivation		After sleep deprivation		t(31)	p
	Mean	SD	Mean	SD		
Depression	14.56	9.67	11.71	9.87	2.137	<0.05*
Anxiety	15.62	5.89	15.50	6.31	0.125	0.902
Anger	15.06	11.16	14.71	10.27	0.297	0.768
Confusion	9.96	5.18	9.37	4.49	1.193	0.242
Fatigue	9.90	6.73	12.12	5.71	-2.189	<0.05*
Vigor	16.28	4.80	14.37	4.89	2.376	<0.05*
POMS total	48.84	34.67	49.06	30.37	-0.058	0.954
DES	9.15	9.15	13.76	14.65	-2.362	<0.05*
WBSI	41.81	11.25	38.37	14.82	2.368	<0.05*
TSH	1.46	0.84	2.14	1.18	-4.708	<0.05 **
T_4	1.05	0.19	1.13	0.13	-2.660	<0.05*
T_3	3.09	0.05	3.34	0.063	-3.541	<0.05 **
DHEA-S	3.06	103.26	3.44	92.98	-2.216	<0.05*
Cortisol	12.44	4.58	15.19	17.90	-0.876	0.388
Cortisol/ DHEA-S rate	29.58	20.03	29.63	13.41	-0.019	0.985

*: $p < 0.05$; **: $p < 0.01$
SD: standard deviation; POMS: profile of mood states; DES: dissociative experiences scale; WBSI: White Bear Suppression Inventory; TSH: thyroid stimulating hormone; DHEA-S: dehydroepiandrosterone-sulfate

tistically significant ($p < 0.05$). There was a positive correlation between cortisol and other blood parameters, which was not statistically significant.

DISCUSSION

In this study, 32 healthy individuals were put through total sleep deprivation to study the effect of sleep deprivation on mood states, dissociation, and thought suppression as well as changes in cortisol and DHEA-S levels and thyroid functions that are known to affect mood states and reveal circadian release.

The result of our study showed a significant decrease in the subscale predicting depressive complaints in healthy individuals following sleep deprivation. In addition, sleep restriction led to a decrease in vigor and activity levels while increasing fatigue levels. It caused an insignificant decrease in the "confusion-bewilderment" subscale, thereby indicating confusion.

The sleep deprivation application provides a controlled regulation in the sleep-wake rhythm. In addition to preclinical and in vivo studies, clinical studies also show that sleep deprivation increases serotonin, noradrenalin, and dopamine neurotransmission, increases thyroid hormone levels, and affects targets in treatment of mood state disorders such as glutamate (18,19,20). The results indicating an improvement in the depressive mood of healthy individuals following sleep deprivation showed that, individual psychobiological differences in endogenous characteristics, diurnal characteristics, and sleep-wake pattern can be the predictors for the response to the treatment (4).

Table 2. Correlation between changes in mood, dissociation, thought suppression and hormonal changes occurring in healthy individuals following total sleep deprivation

	Depression	Anxiety	Rage	Confusion	Prostration	Vigor	Mdp Total	DES	Sea bear	TSH	sT ₄	sT ₃	DHEA-S	Cortisol	DHEA-S Cortisol
Depression	I	0.24	0.24	0.19	-0.13	-0.12	0.51**	-0.49**	-0.08	-0.04	-0.40*	0.26	-0.61**	-0.18	-0.04
Anxiety	0.24	I	0.49**	0.11	0.24	-0.37*	0.67**	0.07	0.20	0.13	-0.06	0.21	-0.12	-0.05	-0.04
Rage	0.24	0.49**	I	0.13	0.66**	-0.55**	0.84**	0.21	0.13	0.11	0.26	0.25	0.14	-0.14	0.02
Confusion	0.19	0.11	0.13	I	0.12	-0.30	0.36*	-0.12	0.04	0.00	0.16	0.26	0.01	0.19	-0.32
Prostration	-0.13	0.24	0.66**	0.12	I	-0.61**	0.64**	0.50*	-0.01	0.04	0.40*	0.07	0.55**	-0.08	0.11
Vigor	-0.12	-0.37*	-0.55**	-0.30	-0.61**	I	-0.73**	-0.06	0.12	-0.22	-0.22	-0.27	-0.11	0.06	0.13
POMS Total	0.51**	0.67**	0.84**	0.36*	0.64**	-0.73**	I	0.04	0.04	0.11	0.02	0.34	-0.03	-0.13	-0.09
DES	-0.49**	0.07	0.21	-0.12	0.50*	-0.06	0.04	I	0.21	-0.18	0.20	-0.27	0.80**	-0.12	0.41
WBSI	-0.08	0.20	0.13	0.04	-0.01	0.12	0.04	0.21	I	-0.20	-0.04	-0.02	0.24	-0.28	0.11
TSH	-0.04	0.13	0.11	0.00	0.04	-0.22	0.11	-0.18	-0.20	I	0.05	0.46**	-0.11	0.18	-0.42
sT ₄	-0.40*	-0.06	0.026	0.16	0.40*	-0.22	0.02	0.20	-0.04	0.05	I	0.09	0.41*	0.02	-0.01
sT ₃	0.26	0.21	0.25	0.26	0.07	-0.27	0.34	-0.27	-0.02	0.46**	0.09	I	-0.08	0.03	-0.16
DHEA-S	-0.61**	-0.12	0.14	0.01	0.55**	-0.11	-0.03	0.80**	0.24	-0.11	0.41*	-0.08	I	0.00	-0.42
Cortisol	-0.18	-0.05	-0.14	0.19	-0.08	-0.06	-0.13	-0.12	-0.28	0.18	0.02	0.03	0.00	I	0.35
DHEAS/ Cortisol	-0.04	-0.04	0.02	-0.32	0.11	0.13	-0.09	0.41	0.11	-0.42	-0.01	-0.16	-0.42	0.35	I

DHEA-S: dehydroepiandrosterone-sulfate; DES: dissociative experiences scale; POMS: profile of mood states; WBSI: White Bear Suppression Inventory; TSH: thyroid stimulating hormone

The studies indicate a decrease in activity, vigor, and excitability in individuals subjected to a restricted sleep time during consecutive days and an increase in fatigue, sleepiness, anxiety, confusion, bewilderment, and irritability (21), whereas in our one night sleep deprivation study, it has been determined that there is no significant change in tension-anxiety, anger, and confusion. This indicates that cumulative effects of chronic partial sleep deprivation may be different from those of acute deprivation and is important to foresee mood state changes that may occur in depressive patients during treatment.

In this study, a significant decrease following sleep deprivation has been determined in total scores from the WBSI scale applied to evaluate the conscious suppression of thoughts or imaginations that individuals do not want to have and perceive as ego-dystonic. These low scores obtained provide the impression that the tendency or the potential to consciously suppress unwanted thoughts decrease following sleep deprivation. Even though such a decrease looks like a positive finding, it may also indicate the effect of a decreased cognitive function along with increased sleepiness.

A significant increase has been determined in dissociation levels following sleep deprivation in our study. This result conforms with the information obtained from the recent research, thus asserting that deviant sleep experiences are associated with dissociative symptoms.

The research has been focused on subjective experiences involving de-realization, absorption, and amnesia complaints, which are dissociative symptoms and abnormal experiences related to sleep (22,23). It has been observed that sleep loss increases dissociative symptoms in long-term shift workers and in groups with a regularly restricted sleep (24). Impairment in the sleep-wake cycle leads to cognitive errors and is also associated with the susceptibility to daydream about imaginary occupations. It also increases hypnotizability and suggestibility (25,26). In our study, both the increase

in dissociation symptoms following sleep deprivation and the decrease in the ability to consciously suppress thoughts indicate that cognitive function disorders caused by changes in sleep-wake cycle can be caused through dissociation or deteriorations in both areas. This is supported by the fact that individuals with high dissociation scores have higher memory neglect errors; this cannot be explained with decreased working memory capacity and high emotional reactivity, and neglect errors are typical in individuals with high dissociation scores (24,26,27).

In our study, a significant increase in dissociation levels following sleep deprivation can be considered as the result of intrusion of electrophysiological characteristics of sleep into wake consciousness. In addition, because norepinephrine, which is a neurotransmitter regulating excitement and alertness, is associated with dissociative symptoms and people prone to dissociation have cholinergic system hypersensitivity, it can be the biological explanation to increased dissociation levels following sleep deprivation (28,29). Acetylcholine, which is released at the highest level in the cortex during wake and Rapid Eye Movement (REM) periods, and neurons localized at peri-brachial area of pons are responsible for cortical desynchronization, rapid eye movements, and muscle paralysis, which are REM components. Because cognitive and behavioral disorders occur due to on one hand, increased serotonergic and noradrenergic activity following sleep deprivation due to mutual interaction between cholinergic and aminergic neurons, resulting in an improvement in mood states, and on the other hand, cholinergic activity is the regulator of conditions associated with cognitive functions such as consciousness, memory, and attention and is related to motor behaviors during sleep, this can shed light on the underlying neurobiological mechanism. In particular, following sleep deprivation causing dysregulation of neurotransmitters such as acetylcholine, serotonin, and norepinephrine, duration of sleep stages that will also occur electrophysiologically and intrusion of failures in transition between such stages and sleep-wake into consciousness may have contributed to

the occurrence of confusion, attention deficit, and dissociative symptoms. Therefore, this study which has a clinical aspect must be supported with neurobiological and electrophysiological studies.

In this study, it has been found that sleep deprivation does not lead to a significant change in cortisol levels of healthy individuals. Conflicting results obtained from former studies also show that a definite result cannot be provided (30). On the other hand, it has been shown that sleep deprivation is perceived as a stressor and thus results in a slight activation in the HPA axis and an increase in plasma concentrations of glucocorticoids (10). In addition, a significant increase has been observed in DHEA-S levels following sleep deprivation. It is known that DHEA-S levels increase due to acute stress in healthy individuals. Preclinical and clinical studies provide indirect evidences that DHEA-S levels and DHEA-S/cortisol ratio can have an important role in the regulation of the effect of stress (31). Clinical studies show that peritraumatic symptoms of dissociation is a risk factor for the development of post-traumatic stress disorder, and these symptoms are positively associated with glucocorticoid release induced by stress (32). It is thought that increased DHEA-S/cortisol ratio can be protective against the negative effects of stress (31). It has been determined that DHEA-S/cortisol ratios during stress are significantly higher in individuals with lesser dissociative symptoms and higher performance (33).

Our study has shown that DHEA-S levels have a positive correlation with DES scores and a negative correlation with depression scores. It has been concluded that lower depression scores in individuals with increased DHEA-S levels following sleep deprivation can be because of the high DHEA-S's protective effect against the negative effect of stress and the improvement in cognition.

In this study, significant increases have been observed in TSH, fT_3 , and fT_4 levels following sleep deprivation. Certain studies state that thyroid function can be predictive in response to sleep deprivation and show that there is an increase in TSH following sleep deprivation (34). Because it shows that thyroxin (T_4) safely elongates clinical response occurring because of sleep deprivation, it strongly supports the relation of antidepressant effect and changes in thyroid functions (35). Similarly, our study is intended to define the biological indicators serving to improve depressive mood following sleep deprivation and the increase in T_4 levels seems to provide this result. As stated before, the increase in T_4 levels following sleep deprivation in our study has been found to be correlated with the decrease in subscales predicting depressive mood. If this result is interpreted correctly, it can guide to determinants studied for the treatment of sleep deprivation in the former studies.

Because individuals have not been evaluated for their morning and evening chronotypes, it is the main limitation of this study. Besides, it constitutes a preliminary study for research that can evaluate the effects of sleep deprivation in neurobiological terms and new research is required to be conducted in this respect.

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REFERENCES

- Richard L, Harper R, Hobson J. Cardiovascular Physiology: Central and autonomic regulation. Principles and practice of sleep medicine. 4. Baskı, Kryger MH, Roth T, Dement WC, WB Saunders Company; 2005, s.192-202.
- Wirz-Justice A, Van den Hoofdakker RH. Sleep deprivation in depression: what do we know, where do we go? Biol Psychiatry 1999; 46:445-453. [CrossRef]
- Wu JC, Bunney WE. The biological basis of an antidepressant response to sleep deprivation and relapse: review and hypothesis. Am J Psychiatry 1990; 147:14-21. [CrossRef]
- Selvi Y, Gulec M, Agargun MY, Besiroglu L. Mood changes after sleep deprivation in morningness-eveningness chronotypes in healthy individuals. J Sleep Res 2007; 16:241-244. [CrossRef]
- Agargun MY, Kara H, Ozer OA, Selvi Y, Kiran U, Ozer B. Clinical importance of nightmare disorder in patients with dissociative disorders. Psychiatry Clin Neurosci 2003; 57:575-579. [CrossRef]
- Giesbrecht T, Merckelbach H. Subjective sleep experiences are related to dissociation. Pers Individual Dif 2004; 37:1341-1345. [CrossRef]
- Durmer JS, Dinges DF. Neurocognitive consequences of sleep deprivation. Semin Neurol 2005; 25:117-129. [CrossRef]
- Killgore WD. Effects of sleep deprivation on cognition. Prog Brain Res 2010; 185:105-129. [CrossRef]
- Lim J, Dinges D. A Meta-Analysis of the impact of short-term sleep deprivation on cognitive variables. Psychol Bull 2010; 136:375-389. [CrossRef]
- Agargun MY, Besiroglu L. Neuroendocrine and behavioral correlates of sleep deprivation: A synthesis of neurobiological and psychological mechanisms. Cardinali DP, Ranti-Perumal SR, editörler. Neuroendocrine Correlates of Sleep/Wakefulness içinde. I. Baskı, New York, Springer; 2006; s.413-422.
- Barret-Connor E, von Muhlen D, Laughlin GA, Kripke A. Endogenous levels of dehydroepiandrosterone sulfate, but not other sex hormones, are associated with depressed mood in older women: the Rancho Bernardo Study. J Am Geriatr Soc 1999; 47:685-691.
- Kuhs H, Farber D, Tolle R. Serum prolactin, growth hormone, total corticoids, thyroid hormones and thyrotropine during serial therapeutic sleep deprivation. Biol Psychiatry 1996; 39:857-864. [CrossRef]
- Blagrove M, Akehurst L. Personality and the modulation of effects of sleep loss on mood and cognition. Pers Individ Dif 2001; 130:819-828. [CrossRef]
- Yargıç Lİ, Tutkun H, Şar V. Validity and reliability of the Turkish version of the dissociative experiences scale. Dissociation 1994; 8:10-12.
- Ağargün MY, Beşiroğlu L, Kiran ÜK, Kara H, Ozer OA. Beyaz Ayı Supresyon Envanteri'nin Geçerlik ve Güvenilirliğine İlişkin Bir Ön Çalışma. Türk Psikiyatri Derg 2004; 15:282-90.
- Mc Nair D, Lorr M, Droppleman L. Profile of Mood States manual, Educational and industrial Testing, I. baskı, San Diego. 1981.
- Selvi Y, Gulec M, Aydın A, Besiroglu L. Psychometric Evaluation of the Turkish Language Version of the Profile of Mood States (POMS). J Mood Dis 2011; 1:152-161.
- Benedetti F, Barbini B, Colombo C, Smeraldi E. Chronotherapeutics in a psychiatric ward. Sleep Med Rev 2007; 11:509-22. [CrossRef]
- Parekh PI, Ketter TA, Altshuler L, Frye MA, Callahan A, Marangell L, Post RM. Relationships between thyroid hormone and antidepressant responses to total sleep deprivation in mood disorder patients. Biol Psychiatry 1998; 43:392-4. [CrossRef]
- Hefti K, Holst SC, Sovago J, Bachmann V, Buck A, Ametamey SM, Scheidegger M, Berthold T, Gomez-Macilla B, Seifritz E, Landolt HP. Increased metabotropic glutamate receptor subtype 5 availability in human brain after one night without sleep. Biol Psychiatry 2013; 73:161-8. [CrossRef]
- Dinges DF, Pack F, Williams K, Gillen KA, Powell JW, Ott GE, Aptowicz C, Pack AL. Cumulative sleepiness, mood disturbance and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night. Sleep 1997; 20:267-277.
- Kucukgoncu S, Aktar E, Erginbas E, Bestepe EE, Calikusu C, Algin F, Erkoç S. Relationship between dissociative experiences, abnormal sleep experiences, and sleep quality in undergraduate students. Düşünen Adam 2010; 23:77-84. [CrossRef]
- Watson D. Dissociations of the night: individual differences in sleep-related experiences and their relation to dissociation and schizotypy. J Abnorm Psychol 2001; 110:526-535. [CrossRef]
- Giesbrecht T, Smeets T, Lepping J, Jelicic M, Merckelbach H. Acute dissociation after 1 night of sleep loss. J Abnorm Psychol 2007; 3:599-606. [CrossRef]
- Blagrove M. Effects of length of sleep deprivation on interrogative suggestibility. J Exp Psychology Applied 1996; 2:48-59. [CrossRef]
- Giesbrecht T, Merckelbach H. The causal relationship between dissociation and trauma. A critical review. Der Nervenarzt 2005; 76:20-27. [CrossRef]
- Giesbrecht T, Merckelbach H. Dissociative symptoms and sleep. Tijdschr Psychiatr 2006; 48:207-215.

28. Hobson JA. The dream drugstore: Chemically altered states of consciousness. Cambridge, MA: The MIT Press; 2001.
29. Koffel E, Watson D. Unusual sleep experiences, dissociation, and schizotypy: Evidence for a common domain. *Clin Psychol Rev* 2009; 29:548-559. [\[CrossRef\]](#)
30. Kryger MH, Roth T, Dement WC. Principles and practice of sleep medicine, Philadelphia, 5. Baski, WB Saunders Company, 2011, s.291-302.
31. Kimonides VG, Spillantini MG, Sofroniew MV, Fawcett JW, Herbert J. Dehydroepiandrosterone antagonizes the neurotoxic effects of corticosterone and translocation of stress-activated protein kinase 3 in hippocampal primary cultures. *Neuroscience* 1999; 89:429-436. [\[CrossRef\]](#)
32. Otis C, Marchand A, Courtois F. Peritraumatic dissociation as a mediator of peritraumatic distress and PTSD: a retrospective, cross-sectional study. *J Trauma Dissociation* 2012; 13:469-77. [\[CrossRef\]](#)
33. Morgan CA 3rd, Southwick S, Hazlett G, Rasmusson A, Hoyt G, Zimolo Z, Charney D. Relationships Among Plasma Dehydroepiandrosterone Sulfate and Cortisol Levels, Symptoms of Dissociation, and Objective Performance in Humans Exposed to Acute Stress. *Arch Gen Psychiatry* 2004; 61:819-825. [\[CrossRef\]](#)
34. Southmayd S, Kasurak P, MacDonald B, Waldron J. Therapeutic sleep deprivation in a depressed patient: prolongation of response with concurrent thyroxine. *Acta Psychiatr Scand* 1992; 86:84-85. [\[CrossRef\]](#)
35. David MM, Owen JA, Abraham G, Delva NJ, Southmayd SE, Wooltorton E, Lawson JS. Thyroid function and response to 48-hour sleep deprivation in treatment-resistant depressed patients. *Biol Psychiatry* 2000; 48:323-326. [\[CrossRef\]](#)